group with lymphocytosis in the spinal fluid. In syphilis of the central nervous system, which is comparatively rare in children, the spinal fluid may give the Wassermann reaction. It also gives the positive Noguchi butyric-acid test. In tuberculous meningitis the Noguchi reaction is negative; the tubercle bacillus is present in nearly 100 per cent. of the cases in the spinal fluid, as was shown by Hemenway. As far as encephalitis is concerned, a number of these cases may be of a parasyphilitic nature and give the Wassemann reaction, or they belong to the cerebral or encephalitic forms of poliomyelitis. It is not unlikely that some cases cannot at present be classified.

The subject of meningism[13] presents serious difficulties and needs further study. The cases belonging to this group must be considered in connection with the underlying infection; the cytology and bacterial findings will vary according to the early or late stage of the disease.

As to poliomyelitis, Flexner and Lewis,[14] Gay and Lucas,[15] and others, have studied the spinal fluid in monkeys and in young children. The result of these studies can best be illustrated by the precise diagnosis of an obscure case from the microscopic examination of the spinal fluid. This was the case of Dr. Frissell: The patient was a young adult in whom all the reflexes were exaggerated, the Kernig sign and Babinski reflex negative, and the neck a trifle stiff. The Widal, and blood culture for the Bacillus typhosus were negative. Leukocytes, 12,000. Physical examination was negative. On the fourth day of the disease, the first lumbar puncture was made. Flexner reported that the fluid was turbid, the sediment consisted of a small number of large lymphocytes, a considerable number of small lymphocytes, and very few polynuclears, and that the clear supernatant fluid gave the Noguchi reaction. The condition of the fluid resembled that seen in inoculated monkeys, just before the onset of the paralysis. On the fifth day of the disease, the reflexes nearly disappeared and there were indications of paralyses of the lower limbs and abdomen. A second spinal puncture showed that the fluid was clear and still contained an excess of lymphocytes. The Noguchi reaction was not so strong. This second fluid resembled that of monkeys after paralysis had set in. The diagnosis and prognosis of this case were made by the most advanced clinical and laboratory investigations.

Nothing is more commendable in clinical medicine than to lay special emphasis upon the thorough observation of the case in hand in all its various clinical aspects. All our methods of observation, both at the bedside and in the laboratory, should be brought to bear on the determination of the given case, just as in criminal law the jury sifts the evidence of the witnesses and takes note of the impression made by the prisoner. The finger-print method of identification seems at present, however, destined to become the scientific test which will outweigh the sum of all the circumstantial evidence; so in our own field the special diagnosis of disease by laboratory methods is advancing in leaps and bounds. Bacteriological, serological, biochemical tests have opened a great vista in the recognition of disease by scientific methods. In acute poliomyelitis we are dealing with a disease, sudden in onset, short in its course, with symptoms obscure and elusive. Although the attempt to diagnosticate it by the serological method has so far been futile, let us hope that its recognition by a simple scientific laboratory test—not to speak of its prevention, cure, and extermination—will be effected in the not too distant future.

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[13] Johns Hopkins Hosp. Rep., 1904, xii, 370, and Richard Stein, Ibid.

[14] Archives of Pediatrics, 1910, xxvii, No. 2, p. 93.

[15] Archiv. of Int. Med., 1910, cxxix, 542.

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# A CLINICAL CONTRIBUTION TO OUR KNOWLEDGE OF POLIOMYELITIS WITH CORTICAL INVOLVEMENT.[1]

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It can hardly be supposed that the more intensive study of epidemics of poliomyelitis of recent years accounts for the bizarre clinical manifestations of this disorder which are now of frequent report. The situation would seem of more rational explanation on the ground that the isolated atypical forms of the disease were overlooked or disregarded in the past. The bulbar and pontine types are now common and well defined, but the cerebral and especially cortical involvements are still subjects of debate despite the classic and well-known contentions of Strümpell and his school on the subject.

The existence of a true encephalitic type of poliomyelitis is difficult to differentiate clinically as well as pathologically. In the first place, a spastic palsy is not definite proof of cortical or encephalitic involvement, and secondly, when the autopsy has shown cortical or meningeal alteration the findings have been susceptible of explanation upon other causative grounds. It is not sufficient to state that an encephalitic type must show a cortical lesion and present evidences of notable meningeal irritation, resulting in hemiplegia, which later becomes spastic, but the character, onset, and clinical course of such cases must be a matter of careful inquiry before a diagnosis of cortical involvement is possible. Opponents of Strümpell's views no doubt hold too rigidly to a true clinical picture of poliomyelitis itself to admit of many of the bizarre symptoms which must obtrude themselves into the make-up of the clinical syndrome of the cerebral type of the disease. A mere report of a sequential palsy, flaccid or spastic, is susceptible of varied interpretations in the individual case. Many times the apparently associated spastic-flaccid states reported are but instances of flaccid palsy with contractures in normal groups of muscles. Taking the matter by and large, there can be no doubt that the few and infrequent case reports of Strümpell's type show that polio-encephalitis in its typical form is seldom encountered in epidemics of acute poliomyelitis and perhaps occurs only in certain epidemics. Thus, in the Report of the Collective Investigation Committee on the New York Epidemic of 1907, on Epidemic Poliomyelitis,[2] it is stated that "while the committee do not doubt the rare occurrence of the cerebral (cortical) type of the disease, no undoubted case of this form was reported and in not a single family was it stated that one child was affected with the acute spinal form of the disease and another with the acute cerebral form. Strümpell's hypothesis of the analogy between polioencephalitis and poliomyelitis is still in need of strict clinical and anatomical proof. The cases previously reported by Moebus, Pasteur, Bucelli, and the doubtful ones of Hoffman and Strümpell are the only cases that offer any support to Strümpell's contention."

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[1] Read before the New York Neurological Society, October 3, 1911.

Even when we consider the less debatable form, the meningeal type, the subject is not less complicated and is equally difficult to demonstrate in the epidemic disease. It is very difficult to exclude the other kinds of meningitis, not alone epidemic cerebrospinal meningitis, but also tubercular meningitis.[3] Meningeal irritation in acute poliomyelitis is not necessarily an initial symptom, but may develop after some days of malaise, etc. Epidemic cerebrospinal meningitis may occur at the same time and place as epidemic poliomyelitis and must, therefore, be excluded. Even a very wide questionare study of epidemics by Zappert[4] failed to establish a complete picture of any meningeal form. He cites 4 cases supplied by this correspondence study. In one family a child, aged eight years, died of meningitis, while one, aged four years, developed a mild case of monoplegia (left leg), and one, aged three years, a paraplegia following what was apparently a severe gastroenteritis. In another household attack, one child developed a paraplegia, its sister a meningitis which ended in recovery. In a third family, two children developed mild transitory meningeal symptoms. Of more interest was a fourth family episode. One child developed meningeal symptoms from which it recovered, but a monoparesis remained (right leg dragged in walking). A brother also developed a meningeal syndrome and temporary right hemiplegia resulted.

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[2] Nervous and Mental Disease Monograph Series, 1910, No. 6, p. 53.

[3] Koplik, The Cerebral Forms of Poliomyelitis and their Diagnosis from Forms of Meningitis, Amer. Jour. Med. Sci., June, 1911, No. 471, p. 788.

[4] Epidemic of Acute Anterior Poliomyelitis, Jahrb. f. Kinderheilkunde, 1910, Band lxxii, S. 107.

While most of us are, therefore, prone to admit, both from clinical as well as pathological evidence, that meningitis may *co-exist* with acute epidemic poliomyelitis in the same subject, an *exclusively* meningeal type appears to be doubtful. If one looks for proof of encephalitis in epidemic poliomyelitis by the presence of cerebral diplegia alone, classic instances of the association are even more rare than the pure meningeal forms just considered. Most writers upon the subject have heretofore confined themselves to this rather rigid test of a pure encephalitic type. If, however, the latitude of inquiry is enlarged to permit of a hemiplegic type also, as well as certain cerebral symptoms known to be cerebral or cortical in origin (such as choreas, athetoses, aphasia, and convulsions), data are not so difficult of attainment. Thus, in the Swedish epidemic of 1905, Wickman saw no cases of spastic hemiplegia, but other symptoms were recorded which pointed to a cerebral localization. Thus, a boy, aged five years, had the usual febrile onset with violent spasms. He could not speak or leave his bed. The spine was somewhat rigid and tender. The head was bent backward, the hands and arms were weak. He was unable to stand without aid. In about a week he had improved greatly, but still dragged the left leg. The knee-jerks were heightened. In this case the motor aphasia certainly showed a cerebral lesion. A medullary location could be excluded. The heightened knee-jerks also spoke for cerebral localization. The usual atrophy of poliomyelitis was absent. The presence of meningeal symptoms suggested a meningo-encephalitis. The lesion must have been trivial, as the patient entirely recovered. Lunggren also saw a patient with motor aphasia in this same epidemic. Bohmann also saw two brothers, one of whom had typical poliomyelitis. The other, aged one year, had a severe right unilateral convulsion followed by paralysis of the whole right side. The child quickly recovered. The case was an unmistakable cerebral and probably a meningo-encephalitic form of epidemic poliomyelitis. However, no author has apparently seen the full development of the typical syndrome of poliomyelitis associated with spastic paralysis, which further illustrates the necessity of broadening the clinical picture if we are to gain records of cortical types of poliomyelitis. In autopsies, however, acute encephalitic and cortical foci in typical epidemic poliomyelitis are of rather frequent report. Redlich found hyperemia of certain cortical vessels and round-celled infiltration in the basal ganglion. Wickman has found the same round-celled infiltration in the cortex of both hemispheres. They were numerous and disseminated. The same lesion was also present in the central ganglia. In another case cited by Wickman no infiltration was present, but there were areas of hyperemia and slight transudation with abnormal richness in nuclei. It must be said, however, in nearly all cases the lesions were of microscopic dimensions. In autopsies of adults the coincidence of old poliomyelitic and encephalitic lesions have also been recognized. Clinical data in this connection, while few in number, are of great interest. Flaccid and spastic paralyses have been seen side by side in children of the same family by Mobius, Medin (4 cases), Pasteur, Bucelli, and Hoffmann. In 1899 Williams reported an association of flaccid and spastic paralysis in the same patient. The child was first seen when eleven years old. When only five years she had headache and convulsions followed by right hemiplegia and asphasia. In a little over a year she recovered or at least improved. He found spastic paralysis of the right arm with flexion contracture. This arm was stronger than the left, probably from its constant choreic twitchings. The right leg was smaller than the left. The knee-jerks were normal. Electric reactions for arm and leg muscles were normal save for the peronei, which showed reaction of degeneration. Analogous cases have been reported by Neurath, Calabuse, Negro, Oppenheim, Marie, and Rossi.

Wickman saw an analogous case as far back as 1899. A girl, aged eleven years, showed evidence of an encephalitis in infancy, before she had learned to walk. The legs were weak for a year, but recovered enough so the child could creep about. When the little patient was first seen, at the age of eleven, there was a high degree of paresis of the lower extremities more marked on the left side. She was unable to stand or walk unaided. When assisted to walk the gait was paralytic, not spastic, but flexing one knee brought out resistance. The muscles of legs were atrophic. There was no reaction of degeneration, but the electric reactions were weak. Intelligence was depressed. Left knee-jerk abolished, right increased. Complete analysis of muscular and tendon phenomena points to an association of cerebral and spinal lesions.

One may summarize the cerebral involvement in poliomyelitis on its anatomic-pathological side as follows: There may be a disseminated encephalitis affecting not only the white substance of the pons and crura cerebri, but also the gray matter of the cortex and regions of the central ganglia. The process, in some instances, would seem to originate in the pia, thus furnishing an important document for the fact that in the epidemic disease the palsy or injury may be a cerebral instead of a spinal one. The random and often unilateral character of the lesion in the cortex is a parallel picture to the pure spinal types. The rarity of the cerebral type is accounted for by Wickman on the ground that epidemic poliomyelitis is an ascending disease and, therefore, must tend to involve the bulb before it reaches the brain. However, it must be said that in experimentally induced poliomyelitis in apes, no encephalitis can be induced, thus seeming to contradict this theory of extension of the virus up the cord by means of the lymph spaces. It is, however, impossible to inoculate the ape's cortex or subdural spaces directly. We are, therefore, forced to conclude that the lumbosacral cord is most predisposed and the cortex least so. As before mentioned, a simple spastic state cannot necessarily be ascribed to a cerebral, or at least, a cortical involvement, as not infrequently the bulb shows such extreme involvement that the pyramidal tract at this juncture is often seriously damaged.

Thus, Müller has seen 4 cases of spastic paralysis none of which could be explained by a polioencephalitis of the cortex solely. One of his cases, a child, aged seventeen months, suddenly developed a spastic hemiparesis after a febrile attack. This was explained by a lesion of the pyramidal tracts within the pons as shown by the crossed peripheral facial palsy. In another case there was a spastic state in one lower extremity and a flaccid palsy in the other. In still a third, there was a hypotonic paresis of the leg and a hypertonic paresis of the arm. There can be little doubt that in the majority of cases with spastic palsies the lesions inducing the same are at the bulbo-spinal level rather than cortical. A very few cases, however, are on record, as reported by Müller and Förster, where a disseminated encephalitis must have been the predominant lesion. I am not aware, however, that any one has reported an exclusive encephalitic lesion in a case of undoubted clinical polioencephalitis. In other words, the clinical and pathological report have not yet coincided in the one case. If the spastic state is, therefore, not a criterion of cortical involvement and can only be considered presumptive evidence, what other facts must we include to make the lesion definitely a clinical as well as a pathological fact? Obviously, the presence of chorea or athetotic movement cannot be a part of the positive criteria as lesions giving rise to these symptoms may be, and indeed are, most frequently subcortical in origin. True motor aphasia, enduring mental enfeeblement such as idiocy and genuine grand mal epilepsy would seem to make the diagnosis of cortical involvement positive. Of all these phenomena genuine and enduring epilepsy might be definitely considered to make the diagnosis of a cortical lesion most certain. I have, therefore, such a case report.

F. S., aged eighteen years. He suffered greatly from indigestion and constipation as a baby. When aged two years, he had one teething convulsion. When aged nine years, after an accidental fall in the water, he had a single convulsion on attempts at resuscitation. When aged fourteen years, he had a typical attack of poliomyelitis as follows: The day before the paralysis set in he had fever, vomiting, prostration, and a convulsion; the next morning he attempted to walk across the room; he had taken but a few steps when he fell and suffered a complete paralysis of all voluntary muscles. The patient suffered a good deal from pain in the muscles of the lower extremities. He slept poorly, but was not delirious. He remained acutely ill for two or three days, could not sit up for several days and was able to stand only with crutches after two or three weeks. The paralysis improved slowly but steadily from above downward. He used crutches to get about for six months. When I first saw the patient one year ago for his grand mal epilepsy which has occurred regularly two or three times every three or four months since the attack of poliomyelitis, he showed a steppage gait, more marked in the right foot. There was moderate palsy and atrophy in the tibials and peronei of the legs. There were reactions of degeneration and some shortening of both postici. The whole condition was most marked in the right leg. The knee-jerks were barely present, being difficult of attainment on the right. There was a normal big toe reflex.

Here, then, is a case of poliomyelitis and epilepsy associated in the same case. Can it be considered a mere coincidence? It may be urged that the occurrence of a convulsion at dentition would argue against the case being one of epilepsy induced by cortical injury in poliomyelitis at fourteen years of age. When one bears in mind, however, the fewness of eclamptic convulsions in infancy that terminate in epilepsy, not more than 2 per cent., the importance of the attack at dentition is of little moment. Again, the boy having a convulsion the result of asphyxiation at eight or nine, after a prolonged submersion (ten minutes), but shows the predisposition to convulsions upon a sufficient cause presenting itself. The ordinary frequency with which spasm and convulsions are encountered at the outset of all infectious fevers in infancy ought to cause one to expect the same phenomenon more frequently in poliomyelitis, a disease of sudden febrile onset. Even supposing that the epilepsy in our case was but one of this character, the few cases of epilepsy or eclamptic attacks examined at death, the sequel to infective fevers, bear no distinct pathology of the cerebral cortex not seen in the reported cases of cortical involvement in poliomyelitis. I am fully aware that these and other reasons for looking askance on my case will occur to many; but before I had unearthed the specific fever (poliomyelitis) at fourteen years of age I had given my opinion that whatever the febrile movement was or had been caused by, his disease as we now see it was induced at that time and was dependent upon that particular infection. The usual mental stigma of a pure idiopathic epilepsy is not here in evidence. The disease in its classic form appeared promptly and has endured since the severe attack of poliomyelitis. Indeed, the first grand mal attack with bladder incontinence and tongue biting came on *pari passu* with the fever and prostration of the poliomyelitis at its inception.

It would seem that we have strong evidence that this patient suffered a diffuse cortical injury at the onset of the poliomyelitis, leaving organic changes in the cortex producing his epilepsy. I can find no similar case on record. The epilepsy in my case has responded illy to the usual hygienic plan of treatment so beneficial in pure idiopathics. The case is placed on record at this time as a clinical contribution to the possible occurrence of cortical injury in a case of otherwise classic poliomyelitis. The residue of the cortical involvement is shown in an enduring epilepsy, a fairly well-proved cortical disease.

While it must be admitted that general or focal convulsions do occur at the onset of polioencephalomyelitis these convulsions or phenomena are for the most part epileptiform in character, lacking the complete picture of genuine epileptic fits, or, they are confined to the acute onset of the epidemic disease or, at least, to the very few days immediately following the attack. There are no cases I have been able to review in the literature in which the convulsions continued as genuine epilepsy in after years such as I am here recording. It is, of course, quite conceivable that this outcome might be expected to occur, as in my case, but it has not come to my notice. The above explanation and disposition holds good for such cases as those of Müller, Zappert, D'Alloco, and Brorström.

It is true that no two nervous disorders are mutually exclusive and in the natural order of events one might expect a certain but small number of epileptics who also had suffered a poliomyelitis. I have seen or known of but one such case in an experience covering several thousand examinations. It may be of interest to cite briefly the details of this one case:

L. L., male, aged thirty-eight years, suffered from a mild attack of poliomyelitis when aged eight years, which affected the whole right extremity. There is now a wasting of the whole right leg from the hip downward and some shortening has resulted. Slight talipes equinovarus and contractures in tibialis posticus of this same side is in evidence. Left knee-jerk is nearly absent while the right appears nearly normal. Achilles jerk is active on the left side and absent on the right. There is a normal big toe reflex. Reaction of degeneration is present in tibiales and peronei. There was a neurotic family history and alcoholism in the father. Patient's first seizure occurred at eighteen. The attacks were grand mal in type, preceded by no aura and were mostly nocturnal. At first the attacks occurred every two or three months; now they are of weekly onset. There is considerable mental deterioration at present.

The case was admitted to my service at Craig Colony several years ago and is still there, a confirmed epileptic. I am indebted to Dr. Shanahan, Superintendent of the Colony, for the case notes here given.

NOTE.—Since this article was written, Richard Stern[5] has published a study upon the future of poliomyelitic children, and has found two cases of epilepsy developing several years after the initial disease. His material embraces 107 cases of poliomyelitis. This author suggests that so long an interval between the poliomyelitis and the subsequent epilepsy took place that suspicion is justified that the affections were coincidental. No data whatever are added. The cases are identical with my case, L. L., in which epilepsy was sequent to poliomyelitis contracted in infancy and in which the comitial disease appeared ten years after the onset of the acute poliomyelitis.

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# THE VALUE OF NAUHEIM BATHS IN NEPHRITIS WITH HIGH BLOOD PRESSURE.

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DURING 1905 and 1906, while associated with the late Dr. Heineman in his work at Nauheim, I paid particular attention to the blood pressures of patients before, during, and after taking the baths, and found in cases of high blood pressure (170 mm. Hg. and over) resulting from kidney complications, that the warm saline baths had a more beneficial effect than the cooler baths containing carbonic acid gas.

Up until that time I had always shared the general belief that Nauheim baths were absolutely contraindicated in all cases of marked kidney disease, and in the literature on the use of the Nauheim treatment which I have consulted, kidney disease, when mentioned at all, is regarded as a contraindication to the baths.

The first thing mentioned by Heineman[1] under contraindications to the Nauheim treatment is chronic Bright's disease. Richard Greene[2] says: "After numerous trials, carefully watched and carefully conducted, Schott found that almost any form of heart disease was benefited by the treatment he elaborated. I have said almost, because it would manifestly be unsuitable for thoracic aneurysm, and much care would have to be observed in treating cases of extensive aortic insufficiency. Also in cases complicated by arterial capillary fibrosis, and small granular kidney." While Groedel[3] as recently as 1910 expressed the following opinion: "Bei patienten mit ungewöhnlich hohem blutdruck, 190 mm. Hg. und mehr, haben wir zuweilen, gerade infolge der verminderung des blutdruckes, schwindelanfälle gesehen. Meist sind dies patienten mit gleichzeitiger nierenaffektion. Wir warnen deshalb davor, solche patienten zur kur nach Nauheim zu schicken, desgleichen patienten mit sehr grossen aneurysmen."[4]

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[5] Jahrb. f. Psychiatrie, etc., 1911, No. 182.

[1] Medical Record, June 22, 1901.

[2] The Schott Treatment for Chronic Heart Diseases.

These observers, whose wide experience with the Nauheim treatmend lends weight to their statements, voice the general belief on this subject. I have found that nearly all physicians are of the opinion that patients with marked kidney lesions are unsuited to the Nauheim baths. My own experience, however, is at variance with this view, for not only have I failed to find a case of nephritis that has in any way been harmed by taking the baths at the proper temperature, but on the contrary, all of my cases have improved. We know that cold bathing, generally speaking, is harmful in such cases, and the baths as usually employed in Nauheim at a temperature of from 83° to 90° F. (28° to 32° C.) are comparatively cool baths. This fact may explain, in part at least, why patients with kidney disease have, as a rule, actually been made worse by the taking of such baths.

In my work at Nauheim during the past three years I have observed a number of cases referred for the cure, in whom various grades of nephritis were present. All of these cases, without exception, were improved by the use of the mild, warm, saline baths given at or near 95° F. (35° C.). Many of these cases have arrived with systolic blood pressures ranging frome 170 to as high as 230, and in all of them the blood pressure has been reduced and the kidney condition improved, in some cases albumin and casts have entirely disappeared. Some of these patients have returned once, and others twice, and in these cases I have found that the benefits derived from the first cure have been lasting, some of them showing no albumin and casts on their return a year later, while in others the albumin and casts, though present, were found in less amounts than on the first visit, and the blood pressure had remained lower.

The beneficial influence on high blood pressure and albuminuria which I have observed following the use of suitable Nauheim baths is best shown by the following cases:

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[3] Bad Nauheim, 1910, p. 118.

[4] With patients who have unusually high blood pressure, 190 mm. Hg. and more, we have noticed occasional dizzy spells on account of the diminution of the blood pressure. Most of these are patients who have kidney trouble at the same time. We, therefore, warn against sending such patients for a cure to Nauheim, also patients with large aneurysms.